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> D HIS

     (FILE 'HOME' ENTERED AT 13:28:27 ON 20 MAR 2006)

     FILE 'MEDLINE' ENTERED AT 13:28:38 ON 20 MAR 2006
L1          2114 S CYSTATIN?
L2         16185 S MACULAR
L3             4 S L1 AND L2
L4        850961 S DNA?
L5          2014 S TRANSLATION PRODUCTS
L6           376 S L4(P)L5
L7            83 S L6(P)(LEVEL# OR AMOUNT#)

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ANSWER 59 OF 83 MEDLINE on STN

AN 87138305 MEDLINE

DN PubMed ID: 3029177

TI Elevated adenosine deaminase activity and hereditary hemolytic anemia. Evidence for abnormal translational control of protein synthesis.

AU Chottiner E G; Cloft H J; Tartaglia A P; Mitchell B S

NC 1-R01-AI24012 (NIAID)

SO The Journal of clinical investigation, (1987 Mar) Vol. 79, No. 3, pp. 1001-5.
Journal code: 7802877. ISSN: 0021-9738.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 198704

ED Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870406

AB We have investigated the molecular basis of the marked elevation in erythrocyte adenosine deaminase (ADA) activity in a kindred with hereditary hemolytic anemia. Red cell ADA-specific activity was verified to be 70- to 100-fold normal **levels**. Western blots demonstrated a corresponding increase in erythrocyte ADA-specific immunoreactive protein. Analysis of genomic **DNA** revealed no evidence for amplification or major structural changes in the ADA gene. ADA-specific messenger RNA (mRNA) from proband reticulocytes was comparable in size and **amount** to mRNA from control reticulocytes. Translation of proband poly A+ reticulocyte mRNA in a rabbit reticulocyte lysate system and immunoprecipitation of 35S-labeled protein products with anti-ADA antibody yielded a band of approximately 42,000 apparent mol wt that was absent in **translation products** from control reticulocyte mRNAs. These data suggest that the increased ADA activity in red cells in this disorder results from the increased translation of an aberrant ADA mRNA.

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ANSWER 55 OF 83 MEDLINE on STN

AN 88007481 MEDLINE

DN PubMed ID: 3654595

TI Isolation of a cDNA clone for murine catalase and analysis of an acatalasemic mutant.

AU Shaffer J B; Sutton R B; Bewley G C

CS Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany 12201.

NC GM-23617 (NIGMS)
RR7071 (NCRR)

SO The Journal of biological chemistry, (1987 Sep 25) Vol. 262, No. 27, pp. 12908-11.
Journal code: 2985121R. ISSN: 0021-9258.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198711

ED Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19871102

AB We have investigated the genetic control of murine catalase expression by analyzing catalase transcription and **translation products** from the tissues of control (Csa) and acatalasemic (Csb) mouse strains. Csb animals possess nearly normal catalase enzyme activity **levels** in liver, while displaying approximately 20 and 1% of normal activity **levels** in kidney and red blood cells, respectively. Immunoblot analyses of catalase in these tissues have revealed reduced **levels** of immunologically reactive catalase protein in Csb kidney and red blood cells, paralleling the reduction of catalase enzyme activity in these tissues. In order to determine the molecular basis for Csb acatalasemia, we have isolated a cDNA clone for murine catalase and have used this probe to analyze Csa and Csb genomic **DNA** and catalase mRNA. These studies have revealed: 1) no restriction fragment length polymorphisms between Csa and Csb genomic **DNAs**; 2) no differences in the **levels** of Csa and Csb catalase mRNA within a single tissue; and 3) no differences in the sizes of Csa and Csb catalase mRNAs. These observations suggest that the genetic defect that produces the tissue-specific reduction of catalase expression in Csb mice is not due to a marked rearrangement of **DNA** within the Csb catalase structural gene. Furthermore, the Csb mutation does not act at the **level** of gene transcription or mRNA stability, but rather at the **level** of mRNA translation and/or catalase protein turnover.

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